

CHRONIC PERITONITIC ENCAPSULATION OF THE SMALL INTESTINE DUE TO TUBERCULOSIS WITH PROLONGED HIGH JEJUNAL OBSTRUCTION

by

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PERITONEAL ENCAPSULATION of the small intestine is an anomaly in which the whole small intestine is found in the mid-abdomen inside a large sac of normal peritoneum (Lewin and McCarthy 1970). Lewin and McCarthy described in their case a small non-constricting aperture in the sac in the lower right quadrant of the abdomen from which the terminal ileum emerged. Superiorly the sac enclosed the duodeno-jejunal junction. Hardas (1970) reported a case associated with malposition of the gut.

Chronic peritonitic encapsulation is a different disorder in which a globular or ovoid sac encloses the small intestine, but the membrane is thick, though not adherent to the surface of the small intestine loops, which themselves are aggregated, and adherent to each other. Presumably this may rarely be the result of chronic peritonitis in a patient with congenital peritoneal encapsulation, but it may more usually be the consequence of the laying down of an inflammatory membrane on the surface of a hitherto normal small intestine. In either case it may be tuberculous, but in many cases no aetiology can be determined. Albot *et al* (1970) have described a case in which jejuno-ileitis of unknown origin led over six years to encapsulating peritonitis with high jejunal obstruction and death. There was no histological evidence of Crohn's disease, nor any of tuberculosis. Albot's case was explored in the second year and the peritonitis was seen in the more acute stage when there was not yet a capsule. Pezzi (1967) described two cases of encapsulating peritonitis with high jejunal obstruction. Both were explored and both survived. There was no histological evidence of any particular disease. However negative histology may not necessarily exclude tuberculosis.

CASE REPORT

The patient, a single woman, aged 34 at the onset of this illness, had been admitted to the Royal Victoria Hospital 14 years before for pleural effusion. Mycobacterium tuberculosis was isolated from the pleural fluid at that time. She made a good recovery from the pleurisy. The present admission was for a continuous fever which had lasted ten days. In spite of the high fever she did not look very ill. There were in the beginning no helpful physical signs. Haemoglobin was 66 per cent. Serum iron was 37 micrograms per cent. E.S.R. 72 mm/one hour. There was an occasional little dry cough. There was no sputum. The chest X-ray showed a small discrete soft shadow in the left upper zone of uncertain significance. Because the fever continued and there seemed no likelihood of establishing a certain diagnosis, anti-tuberculous drugs were begun on the 7th hospital day. Thereafter the fever declined slowly and she became afebrile on the 28th hospital day.

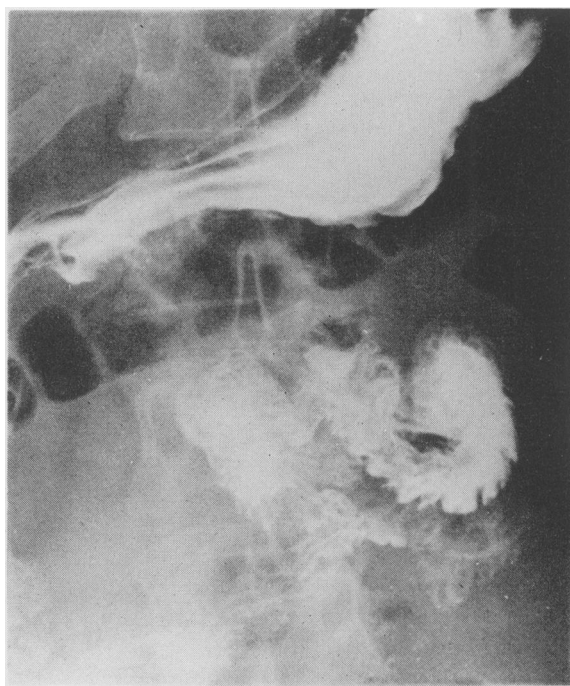


FIG. 1. *Elevation of the stomach and transverse colon by the pseudo-cyst of encapsulating peritonitis. The barium is passing freely into and through the duodenum and upper jejunum.*

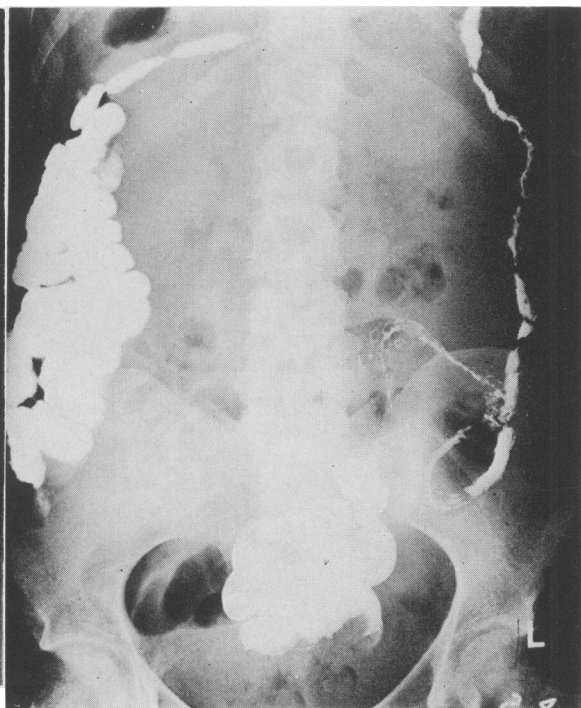


FIG. 2. *The colon is displaced to the periphery of the abdomen by the central abdominal pseudo-cyst.*

On the 20th hospital day the abdomen was noted to be a little full or tumid, and not wasted, as the rest of the body was wasted. The haemoglobin had fallen to 60 per cent. On the 21st hospital day the barium meal showed appearances which in retrospect we realise are consistent with encapsulating peritonitis. The stomach is elevated (Figs. 1 and 3) and the colon is displaced to the periphery (Fig. 2) by the central mass, which contains the small intestine, its loops gathered into a central position (Fig. 3). At that time the barium passed freely enough through the small intestine, though there may have been some delay in the terminal ileal loops. There was no dilatation of the upper jejunum or duodenum (Fig. 1).

About the 25th hospital day abdominal pain and vomiting of obstructive type began, and continued, and grew worse. On the 28th day (when the patient was afebrile) X-ray examination with a Gastrografin meal showed dilatation of the duodenum, and the contrast did not pass beyond the 4th part (Fig. 4).

Because of the obstruction the abdomen was explored on the 29th hospital day. A large pseudo-cyst was found centrally, containing the small intestine (Fig. 5). The cyst wall, which was very thick and white, was opened and partially removed, the contained small bowel was seen to be inflamed, red and friable, and the loops were bound together with adhesions. The cyst contained a small quantity of serous fluid. A narrow compressed transverse colon was found encased superiorly in the

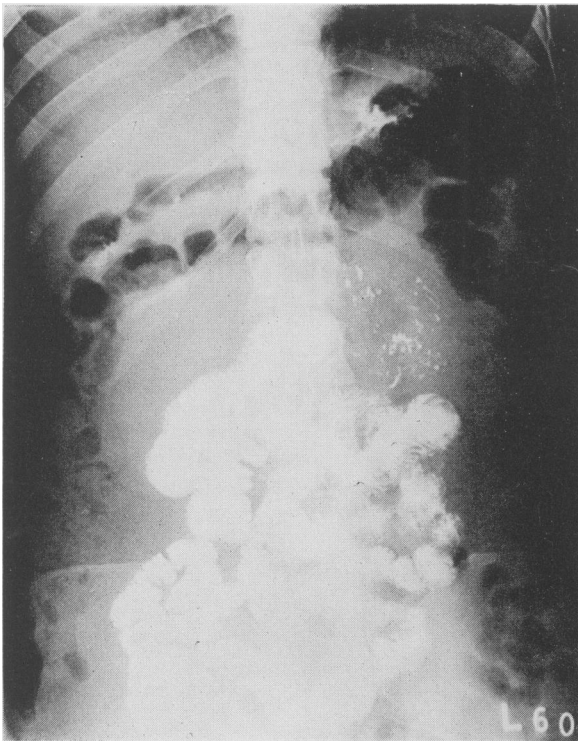


FIG. 3. *The stomach and transverse colon are elevated. The small intestine is gathered into the centre of the abdomen and retained there by the encapsulating pseudo-cyst wall.*



FIG. 4. *Obstruction has occurred. The stomach and duodenum are much dilated. Barium is not passing from the megaduodenum into the jejunum.*

cyst wall. The site of obstruction was found to be thickening in the mesocolon around the duodeno-jejunal region. A surgical by-pass was not possible owing to the thick oedematous jejunum making a gastro-jejunostomy hazardous. Above the cyst the liver and adjacent structures were found to be free of peritoneal thickening.

The obstruction continued unchanged after the operation. The patient was maintained with intravenous nutrition, and the anti-tuberculous drugs were continued. The obstruction continued till the 60th hospital day (it had begun on the 25th), and then the gastro-duodenal aspirations diminished. By the 64th hospital day she was able to eat. At the time the obstruction ceased, after five weeks of intravenous nutrition, staphylococcal bacteraemia with fever occurred. It yielded to treatment with cloxacillin and withdrawal of the intravenous cannulae. However, a septic arthritis led to a stiff right knee.

COMMENT

This note draws attention to encapsulating peritonitis as a manifestation (in this instance) of abdominal tuberculosis. The histological report on specimens of the cyst wall recorded "numerous granulomata with Langhans and foreign-body type giant cells, epithelioid cells and chronic inflammatory cells. In some granulomata

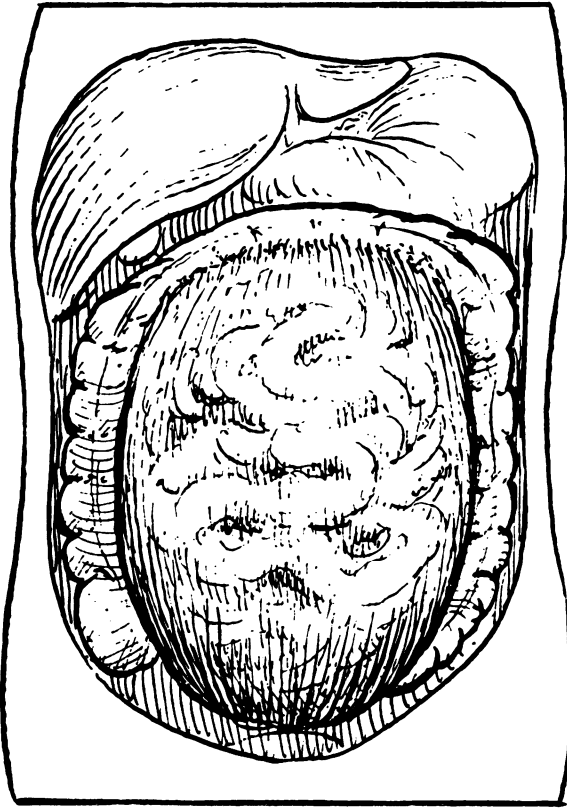


FIG. 5. *Diagram of the appearance of the encapsulated small intestine at operation. The small intestine loops are indicated not because they could be seen through the wall of the unopened sac but to show their situation in the sac.*

there are central areas of necrosis". This seems consistent with tuberculosis. No mycobacteria were seen in Ziehl-Neelsen staining.

In this case anti-tuberculous drugs were of great benefit. When no drug could be taken by mouth, she was treated with the injection of isoniazid as well as the injection of streptomycin. The anti-tuberculous drugs were continued for a year. The patient did well, and is in good health and at work. It is not clear why the obstruction set in when the tuberculous infection was beginning to improve, nor is it clear in what way the obstruction resolved in the end.

High jejunal obstruction seems a common complication of encapsulating peritonitis whatever the aetiology. However, obstruction at the duodeno-jejunal junction by a tuberculous stricture in patients with abdominal tuberculosis, but without encapsulating peritonitis, seems to be well-known in areas where abdominal tuberculosis is still common. Desai and Shah (1970) report nine cases. The patients complained of upper abdominal pain and loss of weight. On X-ray examination there was distension of the duodenum. At operation the stricture at the duodeno-jejunal junction was found.

The possibility of resolution, be it spontaneous or drug-induced, makes necessary resolute, prolonged, intravenous nutrition. It is best to rely for calories on glucose, aminoacid and fructose-ethanol-aminoacid solutions. The serum potassium and

magnesium should be maintained at normal levels. One may use the injection of potassium chloride and dextrose B.P.C. intravenously, and the injection of magnesium sulphate, 50 per cent solution, intramuscularly or intravenously. Hydroxocobalamin and folic acid should be injected, as well as ascorbic acid and the B group of vitamins. A normal iron state, obtained by the injection of Jectofer, helps to support the small intestine mucous membrane, as well as correcting iron-deficiency and anaemia. Infection of the intravenous cannula and thence of the blood stream is a serious danger. The sooner intravenous maintenance can stop, the better.

The X-ray appearance even when there is no obstruction (Figs. 1, 2 and 3) are suggestive enough of encapsulating peritonitis, and may make a pre-operative diagnosis possible. When the case is one of high jejunal obstruction (Fig. 4) encapsulating peritonitis should be considered as a possible cause.

SUMMARY

A case of encapsulating peritonitis of tuberculous origin, complicated by high jejunal obstruction, is described. The obstruction resolved after 35 days of intravenous nutrition. Anti-tuberculous treatment restored the patient to health and full work. The X-ray appearances are helpful in diagnosis, and are reproduced.

ACKNOWLEDGEMENTS

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REFERENCES

- ALBOT, G., PARTURIER-ALBOT, M., RETTORI, R., CAMILLERI, J. P., VEYNE, S., CHRISTOFLE, J. (1970). *Annales de gastro-entérologie et d'hépatologie* **6**, 1.
- DESAI, V. K., SHAH, J. S. (1970). *Advance Abstracts of the 4th World Congress of Gastroenterology*. Copenhagen. The Danish Gastroenterological Association, p. 456.
- HARDAS, K. P. (1970). *British Medical Journal*, **2**, 771.
- LEWIN, K., MCCARTHY, L. J. (1970). *Gastroenterology*, **59**, 270.
- PEZZI, A. (1967). *La Radiologia Medica*, **53**, 1217.